# Successful Transarterial Guglielmi Detachable Coil Embolization of Post-Traumatic Fistula between a Posterior Communicating Artery Aneurysm and the Cavernous Sinus

## A Case Report

Y. CHEN, D-Y. JIANG, H-Q. TAN, L-H. WANG, X-Y. CHEN, J-H. SUN

*Department of Radiology, Second Affiliated Hospital, School of Medicine, Zhejiang University; Hangzhou, China*

**Key words:** posterior communicating artery, carotid-cavernous sinus fistula, detachable coils, aneurysm, intracranial

***Summary***

*We describe a case of a post-traumatic posterior communicating artery (PCoA) aneurysm-cavernous sinus fistula, which is an extremely rare complication of craniocerebral trauma, successfully treated with endosaccular coil embolization via transarterial route. Endosaccular embolization with Guglielmi detachable coils via transarterial route appears to be a feasible, effective and minimally invasive option for the treatment of post-traumatic fistula between the PCoA aneurysm with a small ostia and the cavernous sinus in the subacute phase.*

### Introduction

Posterior communicating artery (PCoA) aneurysm-cavernous sinus fistulae are extremely rare complications of craniofacial trauma [1-3]. Their clinical presentation is similar to that of both direct and indirect spontaneous and post-traumatic Debrun type A carotid-cavernous fistulae (CCF) [4].

The treatment of PCoA aneurysm-cavernous sinus fistulae has not been well described [5]. We describe a case of a post-traumatic PCoA aneurysm-cavernous sinus fistula successfully treated with endosaccular coil embolization via transarterial route. To our knowledge, this is the first report on successful treatment of such fistula using endosaccular Guglielmi detachable coils (GDCs) embolization via transarterial route.

### Case report

*History and Examination*

A 37-year-old man with a severe head injury after a traffic accident was transported to our hospital. He had been previously healthy and had no symptoms consistent with the pre-existing PCoA aneurysm. At the time of patient's arrival at the hospital, his Glasgow Coma Scale score was 5; signs of bilateral rhinorrhea and otorrhea were apparent. Emergency head computed tomography (CT) revealed bilateral frontal lobe contusion and multiple craniofacial fractures involving right petrous pyramid fractures, nasoethmoidal blowout and sphenoid bone fracture. No mass lesion or subarachnoid hemorrhage in the suprasellar cistern were noted. Subsequently, he was admitted to the department of critical care medicine for conservative management.

One month after the injury, polydipsia, hyperdiuresis, a right pulsatile exophthalmos, right conjunctival chemosis, and a retroocular bruit synchronous with the patient's pulse

gradually developed. Laboratory examination revealed urinary volume was 4000-6000 ml/24h, urinary sodium was 458 mmol/24h. He was diagnosed as multiple injury, low sodium syndrome and traumatic diabetes insipidus. In addition, a traumatic carotid-cavernous fistula was suspected. Therefore, he was transferred to the department of neurosurgery for further diagnosis and therapy.

A head non-enhanced CT and CT angiography, a head magnetic resonance examination and cerebral angiography were performed. A non-enhanced CT scan revealed a mass lesion in the right suprasellar cistern (Figure 1A). CT angiography revealed a large saccular aneurysm originating from the PCoA (Figure 1B). Coronal and sagittal reconstructions of enhanced CT images showed the pituitary stalk compression by the the PcoA aneurysm (Figure 1C,D). A T2-weighted magnetic resonance image showed a large saccular aneurysm, dilation of the right ophthalmic vein and the enlarged right cavernous sinus (Figure 2).

An initial angiogram of the right internal carotid artery (ICA) demonstrated that the cavernous sinus rapidly opacified with the engorged superior ophthalmic vein and an aneurysm was in continuity with the cavernous sinus and could not be completely separated from the supraclinoid ICA or the PCoA (Figure 3A,B). Right vertebral angiograms with simultaneous right carotid compression revealed that the cavernous sinus fistula was supplied by a large dome-shaped ruptured aneurysm with a diameter of 10 mm arising from the PCoA (Figure 3C). Thus, a post-traumatic PCoA aneurysm-cavernous sinus fistula was finally diagnosed.

*Endovascular Treatment and Clinical Outcome*

The patient was taken to the angiography suite for endovascular treatment of the aneurysmal cavernous fistula and aneurysm. With the patient under general anaesthesia, using the transfemoral route, a Tracker 10 microcatheter (Boston Scientific/Target, Fremont, CA, USA) was successfully positioned into the aneurysmal sac through a guiding catheter positioned in the right ICA, and one GDC (Boston Scientific / Matrix 360) -10 coil (10 mm x 30 cm) was deposited into the aneurysm. Additional GDC-10 coils (9 mm x 30 cm; 8 mm x 30 cm; 7 mm x 20 cm; 6 mm x 15 cm; 4 mm x 8 cm) were also deposited. Immediately after the procedure, angiography demonstrated complete occlusion of the aneurysm and fistula with preservation of the ICA and the PCoA, without any drainage into the ophthalmic vein (Figure 4A-C). At clinical follow-up three days after the procedure, the patient's hyperdiuresis, exophthalmos and conjunctival chemosis had resolved. There was no filling of the fistula and recanalization of aneurysm on the follow-up angiogram at one year.

## Discussion

Direct CCFs are usually a solitary communication between the intracavernous carotid artery and cavernous sinus as a result of posttraumatic tears of the ICA trunk. However, a small percentage of direct CCFs result from ruptured cavernous carotid aneurysms and systemic connective tissue disorders such as Ehlers–Danlos syndrome [6,7].

Unusual cases, such as traumatic aneurysm of the supraclinoid ICA in association with CCF 8, trigeminal cavernous fistulae [8], CCF produced by ruptured aneurysm of the meningohypophyseal branch and post-traumatic fistula of the PCoA aneurysm-cavernous sinus and the PCoA-cavernous sinus have been reported [1-3,9].

However, to date there is only one other case report in which fistulae developed between the PCoA aneurysm and the cavernous sinus after craniocerebral injury.

Based on the anatomic relationship between the PCoA and its branches, dural branches to the cavernous sinus do not originate from the PCoA. Therefore, the PCoA–cavernous sinus fistula would not occur spontaneously [5]. However, according to the classical description of traumatic arterivenous fistula, the PCoA–cavernous sinus fistula may develop through canalization of a thrombus or through an aneurysmal sac after severe craniocerebral injury [10]. In our case, the vertebral artery injection demonstrated that the PCoA aneurysm initially opacified, and then the cavernous sinus but not the supraclinoid ICA.

Additionally, right ICA injection demonstrated that an aneurysm was in continuity with the cavernous sinus and could not be completely separated from the supraclinoid ICA or the PCoA. These findings are similar to the description of fistula of the PCoA aneurysm-cavernous sinus by Tytle et Al [2], suggesting the formation of

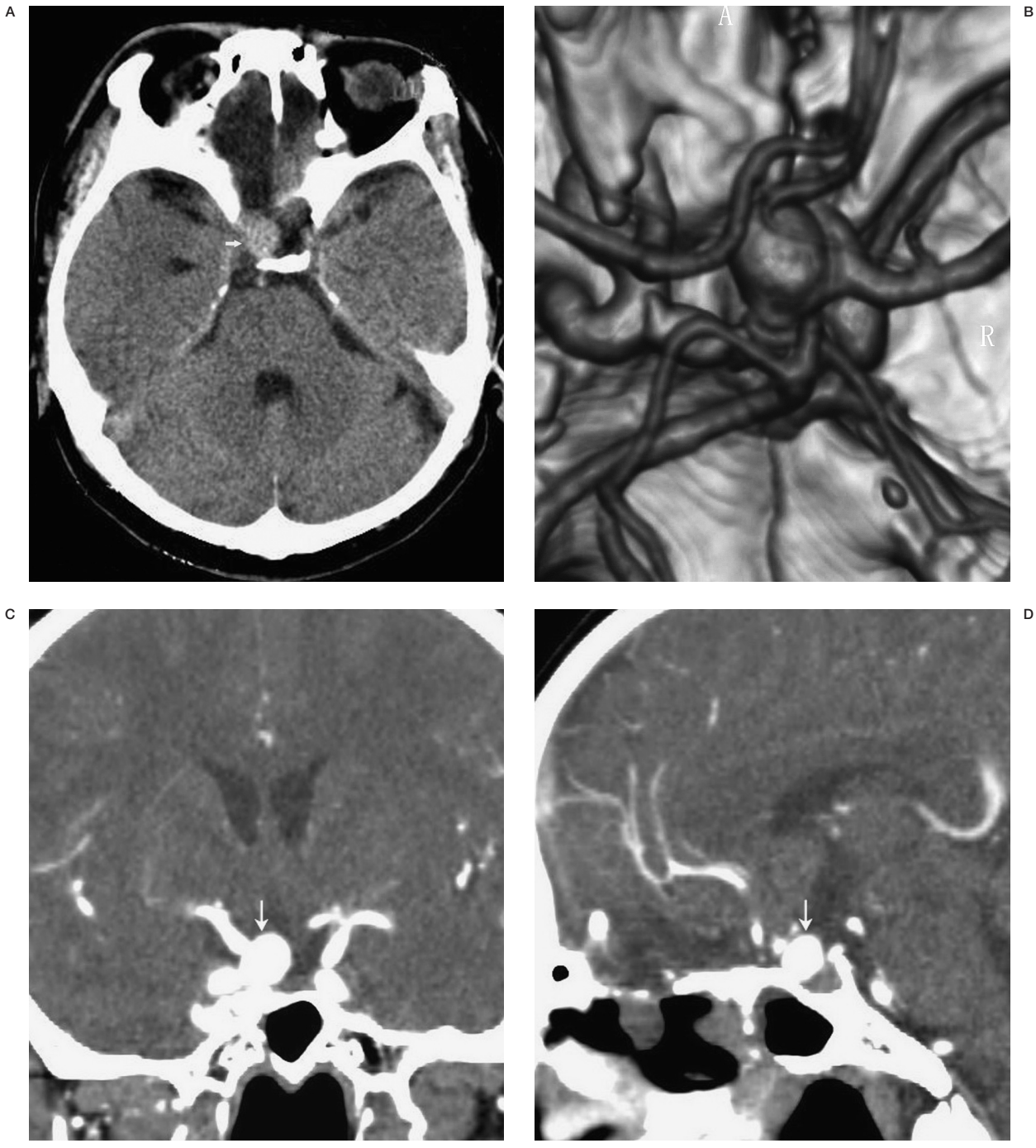



*Figure 1* A) a nonenhanced CT image reveals a mass lesion (arrow) in the right suprasellar cistern. B) Volume-rendering techniques (VRT) CTA reveals a large saccular aneurysm originating from the PcoA. C,D) Coronal and sagittal reconconstructions of enhanced CT images showed the pituitary stalk compression by the the PcoA aneurysm (arrow).

the PCoA aneurysm–cavernous sinus fistula.

An interesting question that arises with the discovery of an aneurysm long after craniofacial trauma is whether the lesion is a pseudoaneurysm secondary to the trauma or an independent preexisting lesion. In our case, although a histopathological study is lacking, we believe that the aneurysm was post-traumatic

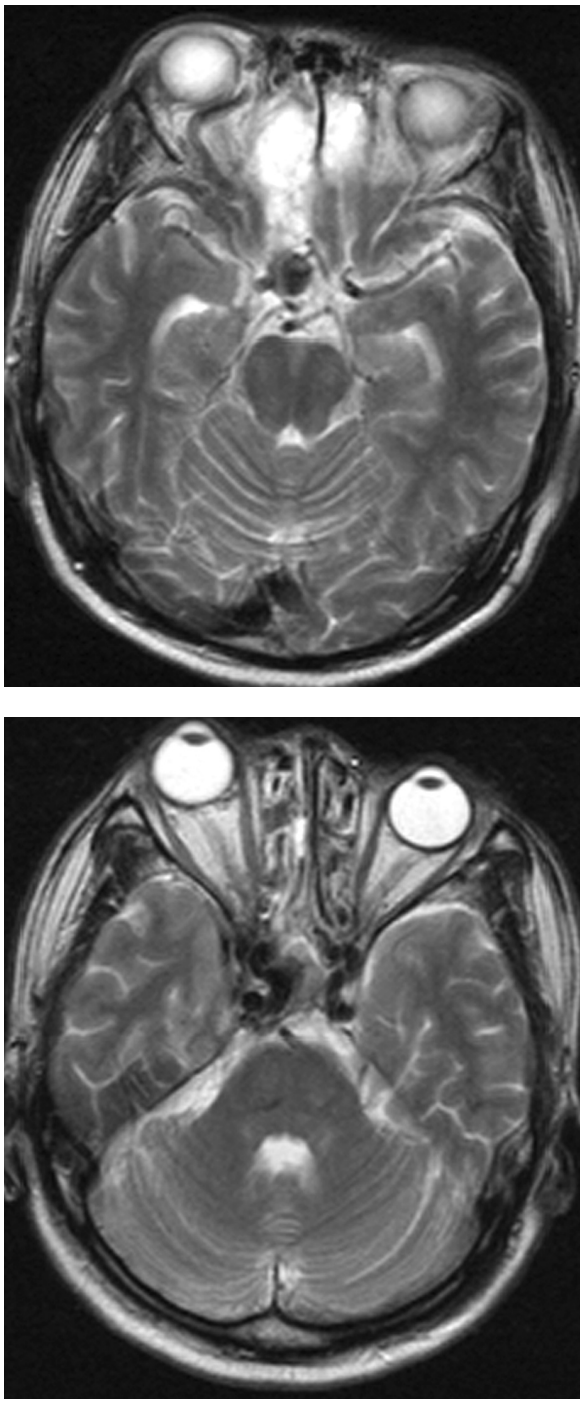



*Figure 2* A,B) Axial T2-weighted magnetic resonance image shows a large saccular aneurysm (A) and abnormal flow void in the enlarged right cavernous sinus proximal (B).

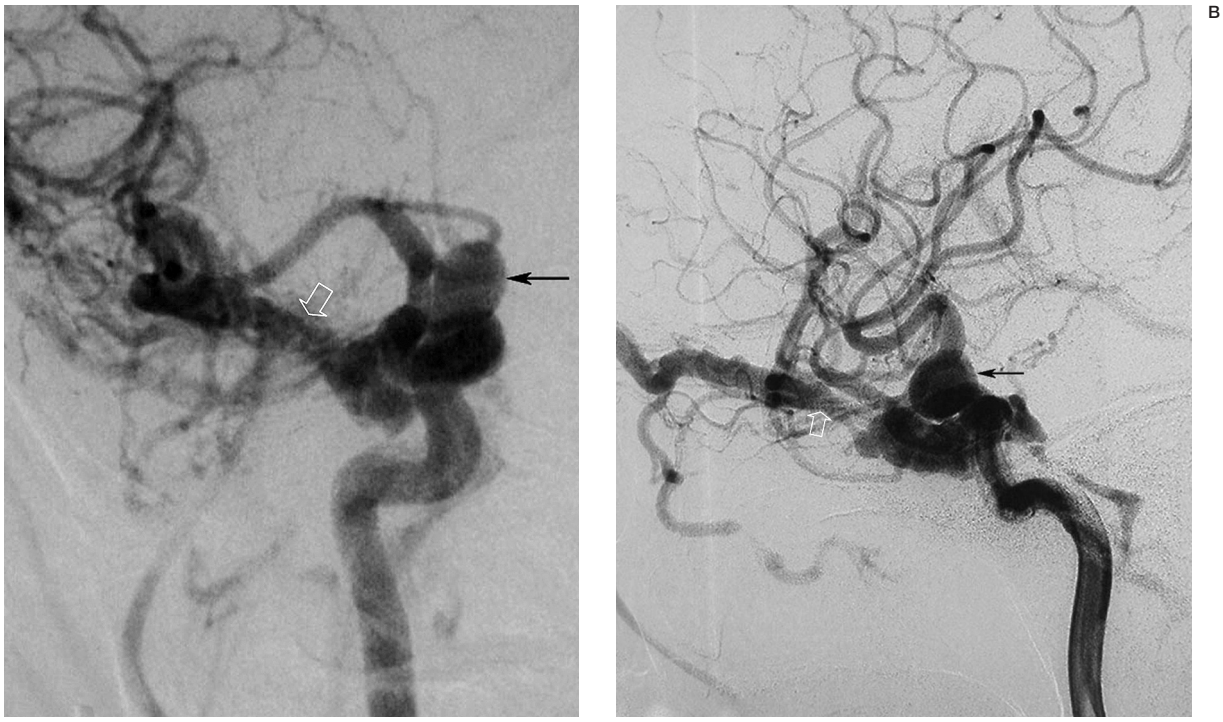
in origin. Our patient had been previously healthy and had no symptoms consistent with the pre-existing PCoA aneurysm. On admission, emergency head CT did not reveal a mass lesion in the suprasellar cistern. However, one month later, a repeated head CT examination and CT angiography revealed a large saccular aneurysm originating from the PCoA. Therefore, we consider that the PCoA aneurysm in our case is a traumatic aneurysm, rather than pre-existing the PCoA aneurysm. In addition, the angiographic characteristics of slow hemodynamics (i.e. delayed filling and emptying of the aneurysmal sac), the absence of a clear aneurysm neck and increases in size over time are indications of a post-traumatic lesion [11-13]. In our case, the traumatic aneurysm enlarged over time and finally ruptured in a delayed fashion so that a fistula between the PCoA and cavernous sinus formed secondary to spontaneous rupture of the aneurysm.

The clinical presentation of CCF arising from the PCoA or PCoA aneurysm is similar to that of CCF from the carotid artery proper, with ocular pain, chemosis, proptosis, disorder of occular movement, visual acuity decrease and a retroocular bruit synchronous with pulse being the common symptoms. Our case presented with hyperdiuresis, low sodium syndrome, and diabetes insipidus besides the above symptoms, which have not been previously reported. In our case, the PCoA aneurysm impinged on the pituitary stalk transporting antidiuretic hormone, leading to decreased transportation and release of antidiuretic hormone and resulting in resorption of water by renal tubule decrease and urinary volume increase.

The treatment options for post-traumatic PCoA-cavernous sinus fistulae have been well documented [3,6]. A transarterial and a transvenous route with GDC have been successfully used to treat post-traumatic PCoA-cavernous sinus fistulae [1,3].

However, thus far the treatment of PCoA aneurysm-cavernous sinus fistulae has not been well described. There is only one case report in which a post-traumatic direct fistula between a PCoA aneurysm and the cavernous sinus was treated by performing proximal anterior clip ligation of the PCoA [2]. In that case, this procedure failed to exclude the fistula because of residual flow from the posterior cerebral artery.

In our case, surgical ligation of the ICA and endovascular therapeutic carotid occulsion was not contemplated because such a procedure not only would have failed to obliterate the fis-




tula, but could have greatly complicated future treatment [2]. Although surgical trapping of the PCoA or surgical clipping may occlude the PCoA aneurysm and obliterate fistulae, both of them are associated with a relative high risk because of recent severe craniocerebral injury and the inherent risk of surgical exposure. Furthermore, surgical trapping permanently sacrifices the potential function of collateral circulation of the PCoA. Therefore, neither of them are an optimal alternative. Transvenous embolization of the cavernous sinus with GDC was contemplated.

This approach may obliterate fistulae, but it leaves the fistulous communication and aneurysm in the subarachnoid space after obliterating the shunting to the cavernous sinus [3]. This approach will be associated with the risk of aneurysmal growth and subarachnoid hemorrhage. Hence, transvenous coil embolization of the cavernous sinus may not be the definitive treatment for such fistula.

Endosaccular coil embolization has become an accepted therapeutic option for the treatment aneurismal carotid cavernous fistulae [14-16]. In our case, although the PCoA aneurysm was not a true aneurysm, the aneurysms had relatively small ostia. In this situation, the delivered coils were stabilized, absorbed the hemody-

*Figure 3* A,B) Right internal carotid angiograms (A, anteroposterior view; B, lateral view) show rapid opacification of the cavernous sinus supplied by the ruptured posterior communicating artery aneurysm, the engorged ophthalmic vein. C) Right vertebral angiogram (C, lateral view) shows the cavernous sinus fistula supplied by the ruptured posterior communicating artery aneurysm.

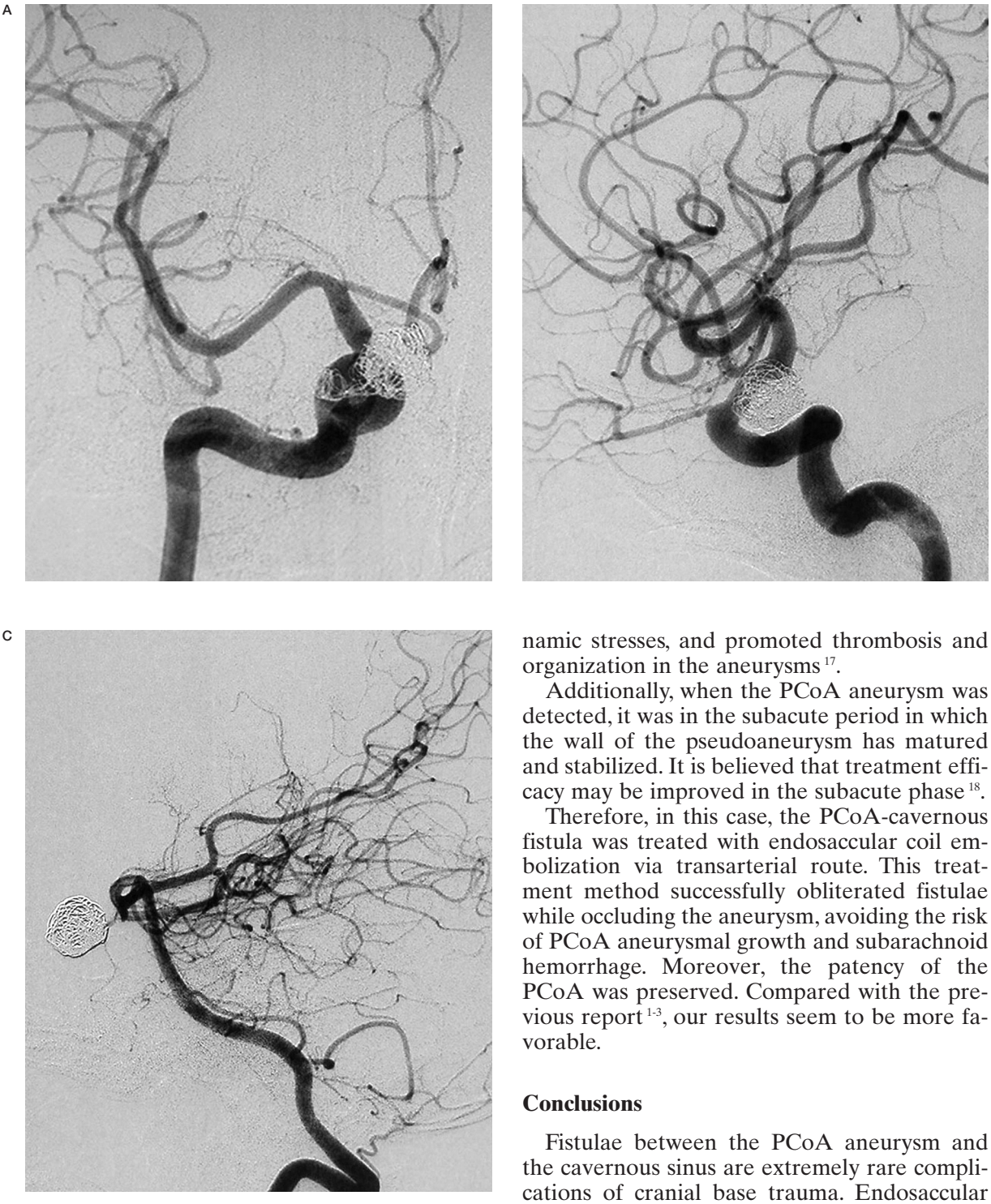



*Figure 4* A,B) Right internal carotid artery angiograms immediately after the procedure (A, anteroposterior view; B, lateral view) demonstrate complete occlusion of the aneurysm and fistula with preservation of the internal carotid artery. C) Vertebral angiogram (C, lateral view) demonstrates complete occlusion of the aneurysm and fistula with preservation of the PCoA.

namic stresses, and promoted thrombosis and organization in the aneurysms [17].

Additionally, when the PCoA aneurysm was detected, it was in the subacute period in which the wall of the pseudoaneurysm has matured and stabilized. It is believed that treatment efficacy may be improved in the subacute phase [18].

Therefore, in this case, the PCoA-cavernous fistula was treated with endosaccular coil embolization via transarterial route. This treatment method successfully obliterated fistulae while occluding the aneurysm, avoiding the risk of PCoA aneurysmal growth and subarachnoid hemorrhage. Moreover, the patency of the PCoA was preserved. Compared with the previous report [1-3], our results seem to be more favorable.

## Conclusions

Fistulae between the PCoA aneurysm and the cavernous sinus are extremely rare complications of cranial base trauma. Endosaccular embolization with GDC via transarterial route appears to be a feasible, effective and minimally invasive option for the treatment of posttraumatic fistula between the PCoA aneurysm with a small ostia and the cavernous sinus in the subacute phase.

## References


1 Kinugasa K, Higashi H, Ohmoto T: Fistula of the posterior communicating artery and cavernous sinus. Am J Neuroradiol 16: 1626-1628, 1995.
2 Tytle TL, Loeffler CL, Steinberg TA: Fistula between a posterior communicating artery aneurysm and the cavernous sinus. Am J Neuroradiol 16: 1808-1810, 1995.
3 Weaver KD, Ewend MG, Solander S: Successful transarterial Guglielmi detachable coil embolization of posttraumatic posterior communicating artery-cavernous sinus fistula: technical note. Neurosurgery 52: 458-460, 2003.
4 Debrun GM, Viñuela F et Al: Indications for treatment and classification of 132 carotid-cavernous fistulas. Neurosurgery 22: 285-289, 1988.
5 Pedroza A, Dujovny M et Al: Microanatomy of the posterior communicating artery. Neurosurgery 20: 228-235, 1987.
6 Debrun GM, Aletich VA et Al: Three cases of spontaneous direct carotid cavernous fistulas associated with Ehlers-Danlos syndrome type IV. Surg Neurol 46: 247-252, 1996.
7 Kanner AA, Maimon S, Rappaport ZH: Treatment of spontaneous carotid-cavernous fistula in Ehlers-Danlos syndrome by transvenous occlusion with Guglielmi detachable coils: case report and review of the literature. J Neurosurg 93: 689-692, 2000.
8 Geibprasert S, Jiarakongmun P et Al: Trigeminal fistula treated by combined transvenous and transarterial embolisation. Acta Neurochir (Wien) 150: 583-588, 2008.
9 Obrador S, Gomez-Bueno J, Silvela J: Spontaneous carotid-cavernous fistula produced by ruptured aneurysm of the meningohypophyseal branch of the internal carotid artery. J Neurosurg 40: 539-543, 1974.
10 Badejo L, Rockwood P: Traumatic arteriovenous fistula of the scalp: case report. J Neurosurg 66: 773-774, 1987.
11 Lee CY, Yim MB et Al: Traumatic aneurysm of the supraclinoid internal carotid artery and an associated carotid-cavernous fistula: vascular reconstruction performed using intravascular implantation of stents and coils. Case report. J Neurosurg 100: 115-119, 2004.
12 Ramos A, Tobio R et Al: Traumatic aneurysm of the internal carotid artery: a late finding presenting as a mass in the sphenoid sinus. Am J Neuroradiol 17: 222-225, 1996.
13 Saito K, Baskaya MK et Al: False traumatic aneurysm of the dorsal wall of the supraclinoid internal carotid artery–case report. Neurol Med Chir (Tokyo) 35: 886-891, 1995.
14 Guglielmi G, Viñuela F et Al: Carotid-cavernous fistula caused by a ruptured intracavernous aneurysm: endovascular treatment by electrothrombosis with detachable coils. Neurosurgery 31: 591-596, 1992.
15 Nishio A, Nishijima Y et Al: Direct carotid-cavernous sinus fistula due to ruptured intracavernous aneurysm treated with electrodetachable coils–case report. Neurol Med Chir (Tokyo) 39: 681-684, 1999.
16 van Rooij WJ, Sluzewski M, Beute GN: Ruptured cavernous sinus aneurysms causing carotid cavernous fistula: incidence, clinical presentation, treatment, and outcome. Am J Neuroradiol 27: 185-189, 2006.
17 Tokunaga K, Kusaka N et Al: Coil Embolization of Intradural Pseudoaneurysms Caused by Arterial Injury during Surgery: Report of Two Cases. Am J Neuroradiol 22: 35-39, 2001.
18 Lempert TE, Halbach VV et Al: Endovascular treatment of pseudoaneurysms with electrolytically detachable coils. Am J Neuroradiol 19: 907-911, 1998.



Dr Ding-Yao Jiang
Department of Radiology
Second Affiliated Hospital
School of Medicine
Zhejiang University, 88# Jiefang Road,
Hangzhou, 310009
People's Republic of China
E-mail: zheerfsk@126.com